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Health Assessment for

WAITE PARK WATER SUPPLY

WAITE PARK, MINNESOTA

DECEMBER 1988

FINAL

Health Assessment for Waite Park Water Supply

I. SUMMARY

Waite Park Water Supply is listed by the U.S. Environmental Protection Agency (EPA) on the National Priorities List (NPL). Volatile organic compounds (VOCs) were discovered in two water supply wells for the City of Waite Park by the Minnesota Department of Health (MDH) in December 1984. An emergency hookup to the water supply of the neighboring city of St. Cloud was established in February 1985. A Limited Remedial Investigation by the Minnesota Pollution Control Agency (MPCA) identified two responsible sources; Electric Machinery (EM) and the Burlington Northern Car Shop (BN). At EM, solvents were found in an area of soil adjacent to the plant. These chemicals have contaminated local ground water and have migrated to the two Waite Park municipal wells. At BN, soil and ground water are also heavily contaminated with a variety of compounds; however, these contaminants do not appear to have migrated to the wells. The BN portion of the site contains two areas of soil contamination that are of immediate public health concern: 1) there is a large pile of sandblast sand containing high concentrations of metals (including lead), and 2) there is an area of buried oily type wastes (which may contain polychlorinated biphenyls; PCBs) that have migrated to the soil surface. These two areas are accessible to anyone who enters the area. There are potential health implications for the residents of Waite Park who may have been using the contaminated water for as long as 15 years, and for children playing in the pile of sandblast sand or contacting the oil seeps.

A treatment system (air stripping) for the water supply was completed in February 1988 and the municipal wells were placed back into service. Results of monitoring confirm that no detectable levels of chemicals remain after treatment.

II. BACKGROUND

A. Site Location and Description

Waite Park is a small suburb on the southwestern border of St. Cloud, Minnesota. It is in Stearns County in central Minnesota.

There are four municipal water supply wells for the City of Waite Park. Wells 1 and 3 are on the BN property and are very productive; Wells 2 and 4 are south of the BN property, but are unproductive and have not been pumped extensively. Municipal Wells 1 and 3 were found to be contaminated with VOCs in December 1984. The wells were taken out of service immediately and an emergency hookup to the St. Cloud water supply was established in February 1985. An investigation by the MPCA identified two sources of contamination; BN, and the former EM, currently Brown Boveri Turbomachinery (BBT), located immediately to the northwest.

The two affected municipal wells are actually on the northeast corner of the BN property. The BN facility is about 200 acres, bounded on the south by Third Street, the east by the St. Cloud Trafficway, the north by an industrial park including BBT, a warehouse and distribution center, and a beverage bottling plant, and on the west by the Sauk River (see attached map). Tenth Avenue runs north-south through the BN facility on the western end. BN has constructed and repaired railroad cars on this site since 1894. The activities on the facility generated waste streams which included oils and greases (found in a lagoon), sandblast sand (found on the surface), calcium hydroxide (CaOH; which occurs in an oil lagoon), solvents (found near a paint building), paints (found on the eastern portion of the site) and PCBs (primarily Aroclor 1254, an inadvertant contaminant of other products or wastes, found in several locations including the oily waste lagoon, the CaOH lagoon, and at two underground tanks). Some oils and fuels used in production were pumped into large underground storage tanks which were overfilled or leaked. BN ceased operations at this facility around 1980 and has donated much of the land to the City of Waite Park. A hockey rink and two baseball diamonds were built on the southwestern portion of the site in 1972 (City of Waite Park Public Works Department, pers. comm.). Several businesses occupy some of the old BN buildings.

The EM contamination may have resulted from mishandled product and wastes (i.e., solvents and paints), and occurs primarily south of the EM building (see map).

The Waite Park Water Supply Site is situated on glacial drift which is generally 75 to 100 feet thick over most of the site. The glacial drift is underlain by Precambrian granite which does not function as a productive aquifer. The depth to the water table averages about 10 feet across the area. The glacial drift is typically separated into an upper and lower aquifer by a leaky confining layer consisting of clay or sandy silt. The confining layer is essentially continuous under the BN property. However, just east of the EM property there is a window at which the confining layer does not occur. The undisturbed ground-water flow direction in the shallow aquifer is radial, towards the river on the west, north and northeast. The flow direction in the deeper aquifer is north. The two Waite Park municipal wells on the BN property have a significant effect on the flow direction when they are pumping, particularly in the deeper aquifer.

A water treatment facility (air stripper) was installed for the city water supply system in Waite Park, and began operation in February 1988. The two municipal wells were brought back into service at that time. The Remedial Investigation (RI) and Feasibility Study (FS) for EM have been completed and a Record of Decision (ROD) is scheduled to be signed by December 30, 1988. The RI for BN has been completed, the FS has not been completed; however, some interim remediation is taking place at the site. A ROD for BN is anticipated for 1989.

The following documents were provided to MDH for review:

Final RI Report for the Burlington Northern Site, August 1988, ERT.

Long Term Water Supply Response Action Final Report, Burlington Northern, May 1988, ERT.
MPCA Limited RI for Waite Park Water Supply, February 1986.
Waite Park Water Supply Study- Final FS Summary Report, April 1986, Rieke Caroll Muller.
Results of Additional Investigation Assessment of Response Action Alternatives- Electric Machinery Site, January 1988 and revised April 1988, Barr Engineering.
Proposed Remedial Action for Contaminated Ground Water at Burlington Northern Site, Waite Park, Minnesota. Office Memorandum from Lisa J. Thorvig, MPCA, Division of Air Quality to Maureen Johnson, MPCA, Site Response, Ground Water and Solid Waste, March 11, 1988.

There was also consultation with the project staff at the MPCA and with the Public Works Department Superintendent for the City of Waite Park. A site visit was also conducted by MDH staff. Information obtained from these sources form the basis for this assessment.

B. Site Visit

A site visit was conducted by MDH staff on August 18, 1988. The MPCA On-Site Inspector was also present. Information obtained during the site visit is incorporated into the text. The City of Waite Park has built a hockey rink and two ball fields on the southwest corner of the BN property. The area immediately north of this is extensively used; there were dirt bike trails, walking trails, litter and debris. In this area the oil and grease has seeped to the surface in several places by the lagoon. The pile of sandblast sand (see Section II. A. for discussion) is easily accessible.

III. ENVIRONMENTAL CONTAMINATION AND PHYSICAL HAZARDS

Chemicals are included in this evaluation based on several considerations. These considerations are as follows: 1) MDH has developed Recommended Allowable Limits (RALs) for contaminants in private drinking water supplies. Chemicals included on this list are those most frequently found in ground water in the state. RALs for non-carcinogens are often taken from the drinking water health advisories published by EPA Office of Drinking Water (ODW). RALs for carcinogens are derived from the potency slopes from EPA Carcinogen Assessment Group (CAG) and reflect an estimated lifetime excess cancer risk of 1 in 100,000. Chemicals found in ground water at concentrations which exceed these RALs are included in this evaluation. 2) Chemicals which do not exceed the RALs may also present a hazard or potential hazard to public health, based on site specific information and professional judgement. In the present case, numerous chemicals were detected in the municipal wells and at very high concentrations in the monitoring wells. There is concern when there are low levels of many chemicals because of the unknown consequences of exposure to mixtures. Future research data on health effects of mixtures of these chemicals may indicate a need for further evaluation. 3) Additionally, monitoring results may be impacted by the variability of glacial formations typical of Minnesota and the imprecise nature of hydrogeologic characterization.

Therefore, some chemicals currently detected at low concentrations may be detected at much higher concentrations in future sampling events. 4) Chemicals in the soil that were present in concentrations that could potentially pose a health hazard were included in this assessment. For these reasons, elimination of these chemicals from the evaluation was not warranted at this site.

A. On-Site Contamination

Soil contamination on the EM property consists of VOCs in the ground behind the EM building (currently the BBT building; see attached map). Most of the contamination was found between 1.5 and 15 feet in depth, although there was some minor surface contamination. Soil tetrachloroethylene (PCE) concentrations were as high as 1,800 mg/kg in several places, soil gas concentrations were frequently over 1,000 ppm for PCE. There were some areas where PCE contamination existed in the surface soil; concentrations were as high as 430 mg/kg with corresponding soil gas measurements of greater than 1,000 ppm PCE. Lower concentrations of trichloroethylene (TCE), xylenes, 1,1-dichloroethylene (1,1-DCE) and 1,2-dichloroethylene (1,2-DCE) were present in a few areas. There are approximately 50 cubic yards of contaminated soil on this site.

Contamination in the soil has leached into the aquifers below the EM site. Maximum concentrations of VOCs measured during the RI in the shallow and deep aquifer are presented in Table 1 (see Section II. A. for discussion).

The BN facility is more complicated. The area west of Tenth Avenue (see map) was used as a disposal area for several waste streams generated by the car repair process. There is a large pile of about 300 cubic yards of sandblast sand in this area; the pile has been there about 10 years (City of Waite Park Public Works Department, pers. comm.). The sand contains high amounts of metals, including arsenic (As), chromium (Cr) copper (Cu), lead (Pb), nickel (Ni), and zinc (Zn). The concentrations of these metals in the sandblast sand is currently being determined. There is a large area of oily soil which contains 1,200 ppb oils and grease (3.3 percent dry weight), acetone (200 mg/kg), PCE (380 mg/kg), and PCBs (570 mg/kg). Other subsurface soils in this area contained 40 mg/kg As, 52 mg/kg Cr, 7,100 mg/kg Cu, 66,000 mg/kg Pb, 78 mg/kg Ni, and 8,300 mg/kg Zn. The contaminated soil is mostly below the surface at depths of 4 to 12 feet, but in some areas of the lagoon the oily substance has seeped to the surface. Some surface soil samples were taken at a discharge point to the Sauk River (see map); these samples contained 3,500 mg/kg Pb and smaller concentrations of the other metals.

To the east of Tenth Avenue there are several areas of oily soils (see map). The oily area at the corner of Tenth Avenue and Third Street contains 104 mg/kg PCBs and 3.8 percent oil and grease. The oily soil to the far east of the site contains no PCBs, but does contain several VOCs (methylene chloride, 1,100 ppb; acetone, 4,700 ppb; fuel oil products such as benzene, toluene, and xylene, 111 ppb; 2-butanone, 49,000, ppb). Also,

sandblast sands with corresponding high concentrations of metals are piled in the far east corner. There is also an area (at the corner of Tenth Avenue and Third Street) of 300 to 500 cubic yards of CaOH which contains high concentrations of some metals. There are several areas where buried or above ground tanks have spilled, leaked or been overfilled and have contaminated the subsurface area immediately around the tank, in many cases to the water table. Most of these tanks contained fuel or lubricating oils.

Ground-water contamination is extensive across the entire BN property. In the vicinity of several underground tanks, there is free product on the top of the water table. The ground water under the area west of Tenth Avenue is also contaminated. There has not been much movement of the contaminants in the ground water and most of the contamination is very localized. Maximums across the entire BN facility are presented in Table 1. As a rule, the contaminants have not migrated very far in the upper aquifer, probably because the hydraulic gradient is very low in this area. There has been some leakage of chemicals into the lower aquifer, but the concentrations are very low, probably because the confining layer is acting to retard vertical movement of the chemicals.

Maximum concentrations of VOCs that were found in the municipal wells (1984-1985) are listed in Table 1.

B. Off-site Contamination

Sampling results indicate that there was no ground-water contamination detected in off-site wells.

Sampling and analysis routinely conducted by the Minnesota Department of Natural Resources (DNR), in conjunction with the MPCA and MDH, shows that fish in the Sauk River below the BN site are contaminated with PCBs. Smallmouth bass contain an average of 0.4 mg/kg total PCBs in the edible fillets (MDH, Health Risk Assessment Section, 1987). Sediment samples taken in June 1988 indicate no significant elevation of PCB levels at or downstream from the BN site. PCB contamination also exists upstream from the site. Further investigation into the source of the PCB contamination is pending. Water samples at a MPCA monitoring station taken over the past three years indicate no elevation in metal concentrations in the water in this area of the river.

C. Physical Hazards

There is much debris left from the operation of the railroad car shop, much of which would constitute a physical hazard to young children who might play at the site. There are piles of railroad ties, scrap metal, broken glass, old machinery and piles of junk all around the yard. Most of the debris is in the BN area east of Tenth Avenue, and this area does not appear to be accessed much by the public. The area to the west of Tenth Avenue contains some scrap piles, but the debris is not as extensive and does not appear to pose a physical hazard.

IV. DEMOGRAPHICS OF POPULATION NEAR SITE

A. Population and Land Use

Waite Park is a suburb to the southwest of St. Cloud, Minnesota. Waite Park has a population of about 4,000 (Public Works Supervisor, pers. comm.). About 8.2 percent of the people in Waite Park are under 5 years of age and 10.5 percent are over age 65 (Census, 1980). The municipal water system has about 750 connections and serves 3,500 people. To the north of the site is an industrial park; several of the companies in the park have lawn watering wells screened in the contaminated lower aquifer. The EM lawn watering well has shown trace levels of the contaminants. Sampling has shown that the other lawn-watering wells are not contaminated. Further north is a high school and a veterans hospital. Both buildings are on municipal water from St. Cloud (MDH, Water Supply and Engineering Section). To the west is the Sauk River, which curves around to join the Mississippi River about 2 miles northeast of the site. To the south is a residential area with some commercial businesses interspersed, some of these residences and businesses have private sand point wells which are not in the path of contaminant movement from the site. The nearest houses are approximately 50 feet from the site. To the east is a commercial and residential area. The St. Cloud water supply is drawn from the Mississippi River about 1.5 miles east of the site and from a ground-water well located near the surface water intake. The well is not in the direction of the plume.

V. EVALUATION

A. Data Needs and Evaluation

1. Environmental Media

The data available for the EM property soil contamination is adequate to conduct a Health Assessment. The extent of ground-water contamination has been adequately defined. The data for soil contamination on the BN site is also adequate. The metal content of the sandblast sand is not known, but is being sampled currently.

2. Quality Assurance/Quality Control (QA/QC)

Data supplied is adequate to conduct a Health Assessment at this site. Sampling techniques and chain of custody were in accordance with the Quality Assurance Project Plan (QAPP). Selected samples were split with MDH, Environmental Research Technology and Twin City Testing (Consulting Laboratories) and variation was determined to be within acceptable limits. Laboratories used for the analysis were EPA Contract Laboratory Program (CLP) Laboratories. Upon examination of the QA/QC information available in the RIs, the analytical techniques are judged to be adequate. The data presented in the RIs are considered by MPCA and MDH to be useful and of high quality.

3. Demographics and Land Use

The demographic information gathered on the site visit and from the Public Works Supervisor for the City of Waite Park is adequate to assess the health implications of this site. If land use in the area were to change, or if private wells were to be installed to the north or northeast of the site, this assessment would not be valid. In addition, if the City of Waite Park were to develop the land given them by BN before cleanup is complete, this assessment would not be valid.

B. Environmental Pathways

1. Ground Water

When the Waite Park municipal wells are pumping, flow is towards the municipal wells in the deep aquifer and water is pulled from the surficial aquifer through the window in the confining layer. This action pulls contaminants from the shallow aquifer into the deeper aquifer. This has caused the municipal wells to intercept contaminants from the EM facility. It appears that a significant portion of the contamination in the municipal wells is actually from the EM facility rather than the BN facility.

At the time of the RI work, the municipal wells were not pumping. This allowed the contaminants to move out radially from the EM facility (with the ground-water flow). MPCA staff estimated that there are three plumes of VOC contaminants from the EM facility (see map); one extending from the most highly contaminated area to the municipal wells, originating in the upper aquifer and moving through the window into the lower aquifer, one moving north from this area in the upper aquifer, and a smaller plume by the railroad tracks in the upper aquifer. Now that the municipal wells are back on-line, it is expected that the wells will continue to draw the contaminants again. Although there are contaminants in the ground water beneath the BN facility, these contaminants are unlikely to be the source of the contamination in the Waite Park municipal wells (based on monitoring data, Section II.A.)

2. Surface Water

The Sauk river, which forms the western boundary of the BN facility, is the only surface water body near the site. The site is flat and has a good cover of grass, bushes and trees near the river. There was little evidence of gullying or eroded material entering the river observed during the site visit. The eastern portion (the portion east of Tenth Avenue) of the BN facility is bounded by ridges formed by the road on the south and the railroad tracks on the north, forming an internal drainage system. The contamination at the EM facility is primarily deeper soil and ground-water contamination which would not affect surface water. Here the site slopes towards the railroad tracks on the south and has a good cover of grass so that drainage would be internal and surface runoff leaving the site would be minimal.

Soil on the river bank which in the past received discharge from a pipe at the BN site was sampled and found to contain heavy metals. Although no sediment samples were analyzed for metals, water samples in the area indicate no increase in concentration of metals in the water. Sampling for PCBs in river sediments indicate no significant contribution of PCBs from the BN site.

3. Soil

Soil samples taken at both EM and BN indicate extensive contamination of this medium. At the EM facility, the contamination is subsurface; MPCA estimates that there is about 50 cubic yards of contaminated soil. At the BN facility, most of the heavily contaminated soil is also below the surface of the ground. The buried tanks and one above-ground tank, which were the source of much of the contamination, are being removed during an interim remediation to reduce further contamination of the aquifer. During the process of tank removal, soil found to be contaminated is removed and is temporarily being stored on plastic liners and covered with tarps. The FS for this site will address further action regarding remediation of soil contamination. The area to the west of Tenth Avenue contains surface oil seeps around the lagoon area and a large pile of sandblast sand (about 300 cubic yards). A resident indicated that the seeps have been there a long time, and that during hot weather the size of the seeps increase.

4. Air

VOCs may volatilize at the EM site into the air above the contaminated soil since soil gas measurements were so high. The sandblast sand present at the BN site is capable of contributing to fugitive dust blown off the site.

Current treatment of the Waite Park Water Supply is air stripping. Stack emissions testing and dispersion modeling (Industrial Source Complex-Long Term; ISC-LT) of the air discharge from the facility was conducted. MPCA and Barr Engineering have also conducted ISC-LT dispersion modeling on emissions from the proposed EM air stripper, assuming 100 percent removal of the contaminants from the water. Maximum ambient air concentrations were estimated to be 0.04 ug/m^3 TCE, 0.11 ug/m^3 PCE, 0.005 ug/m^3 chloroform, and 0.006 1,1-DCE. This information was used to evaluate the potential combined impacts of toxic air emissions from both air stripper facilities at the Waite Park site.

5. Biota

Some fish in the Sauk River and in the Mississippi River at Sauk Rapids (where the Sauk River joins the Mississippi River) are contaminated with PCBs. Smallmouth bass contain an average of 0.4 mg /kg total PCBs in edible fillet and are under a fish consumption advisory (MDH, Health Risk Assessment Section, 1987). Sediment samples upstream and downstream indicate that this contamination is not from the BN site.

C. Human Exposure Pathways

1. Ground Water

Contamination of the Waite Park Municipal Wells 1 and 3 was discovered in December 1984. Residents using the municipal supply were immediately instructed to use water from an alternate source which was available at various locations throughout the city. Emergency hookup to the St. Cloud water supply was established in February 1985. These wells were the only wells that were pumping water at the time, and they were the only ones that had been pumping since 1973. Therefore the water from the contaminated wells was not diluted with an uncontaminated source before distribution to users. MPCA interviews with former employees indicate that soil disposal of solvent and paint waste may have occurred from 1969 to 1977 at the EM facility (Barr Engineering, 1986). Contamination of the wells may have occurred soon after the dumping started. Persons using the municipal water system in Waite Park have been exposed to these VOCs via ingestion of water and via inhalation of chemicals volatilized from the water, and to a much lesser extent, via dermal absorption.

At this time, treatment has been installed on the Waite Park municipal supply system (air stripping). Although water in the wells still contain contaminants, testing results indicate the water after treatment contains undetectable levels of contaminants.

2. Surface Water

Residents of the community indicate that the Sauk River is used for canoeing, swimming and fishing. There is no contribution of PCBs from the BN facility in the Sauk River. Although it is not known if there are metals in the sediments, routine monitoring confirms no increased concentrations of metals in the river water. Therefore, appreciable human exposure to contaminants from this site via this route is unlikely.

3. Soil

Surface soil at the BN facility west of Tenth Avenue is contaminated with sandblast sand and with oil wastes at the lagoon to the west of Tenth Avenue. This contamination is a potential pathway for human exposure. On this portion of the BN facility, much evidence of human activity was observed during the site visit; i.e., dirt bike trails, paths, discarded clothing, litter etc. were in abundance. A resident stated that the sandblast sand pile was used for a dirt-bike jump. It is also in very close proximity to baseball fields and a hockey rink. The contaminated material is certainly accessible, and children playing in the area are at risk of exposure to the metal contaminants in the sand. There is less accessibility to the contaminants on the eastern side of Tenth Avenue because there is little surface contamination, and also, because this portion of the site is less attractive for non-occupational human activity. Sandblast sand on the far east portion of the site may result in some exposure. The contaminants on the EM facility are not accessible; the area has good vegetation and is well fenced.

4. Air

The sandblast sand on the site is capable of contributing to fugitive-dust exposure, and thereby presents a health risk because of the metals associated with the sand. Residences to the south of the site, and people using the recreational facilities south of the site may be near enough to receive this dust, but since the predominant summer wind direction is to the northeast, significant exposure from this route is not likely. Persons who come into contact with the sand are certainly exposed to the dust.

The remediation for the Waite Park municipal water supply is air stripping; the facility is located at the municipal wells. The remediation for the ground-water contamination at the EM facility will also be air stripping. The maximum concentrations of VOCs that may be emitted from these facilities are outlined in the environmental pathways section (see section V.B.4). Nearby residences will not be exposed to concentrations greater that those outlined in Section V.B.4.

5. Biota

There are no organisms which constitute a part of the human foodchain at this site which are contaminated with contaminants from this site.

VI. PUBLIC HEALTH IMPLICATIONS

1. Contaminants of Concern

PCE is readily absorbed into the body via ingestion and inhalation and is deposited into adipose tissue. Dermal absorption at low concentration is probably insignificant (Stewart and Dodd, 1964), although cuts and lesions may allow greater absorption. Most of the absorbed dose is excreted unmetabolized through exhalation, and a small amount is metabolized in the liver to trichloroacetic acid and trichloroethanol which is excreted in the urine. Exposure to high concentrations (100 ppm) has been associated with neurological toxicity in humans (EPA, 1986). These effects are also seen in animals. Liver and kidney effects have also been noted after long periods of exposure (EPA, 1986). PCE has not been found to be teratogenic. Mutagenicity studies of PCE have produced negative or inconclusive results, although a reactive metabolite of PCE, PCE epoxide, has been shown to be mutagenic. Human epidemiologic studies have been inconclusive because most workers exposed to PCE are also exposed to other solvents. Animal studies by NTP (1986) and NCI (1977) have shown positive carcinogenic responses. PCE is classified by CAG as a class B2 carcinogen (probable human carcinogen; EPA, 1986) by both the oral and inhalation routes of exposure. CAG has estimated that lifetime ingestion of water (2 liter/day) containing 6.7 ug PCE/liter would present an increased cancer risk of 1 excess cancer per population of 100,000. However, because the classification of PCE is controversial ODW has set Lifetime Health Advisory at 10 ug/l for non-carcinogenic endpoints (EPA, 1987), based on a reference dose of 0.0143 mg/kg/day (drinking water equivalent level of 500 ug/l). MDH uses 6.7 ug/l for its RAL for PCE.

TCE is also readily absorbed into the body via ingestion and inhalation. Like PCE, most of the dose is exhaled unchanged, but unlike PCE, most of the absorbed dose is metabolized to trichloroethanol, trichloroethylene-glucuronide, trichloroacetic acid, and other minor metabolites. Elimination is slow. TCE also distributes to fat. Primary targets for TCE are the central nervous system (CNS), liver, kidney and hematological system (ATSDR, 1988). Immune effects have been noted in animals at chronic doses of 18 mg/kg (Sanders et al., 1982), and hematological effects at 50 mg/kg (Nomiyama et al., 1986). TCE metabolites have been shown to be weakly mutagenic. Reports of human exposure to TCE are not useful in evaluating carcinogenic potential of this compound; all of the studies are controversial or have design flaws. However, TCE was found to induce hepatocellular carcinoma in mice (NCI, 1976), and liver neoplasms in mice (NTP, 1982). Because of the positive animal studies, CAG designates TCE as class B2 (probable human carcinogen, EPA, 1986). Further support for classifying TCE as a probable human carcinogen comes from studies that indicate that the metabolism is qualitatively similar in animals and humans. CAG has estimated that lifetime ingestion of water (2 liters/day) containing 28 ug TCE per liter of water would present an increased cancer risk of 1 excess cancer per population of 100,000 (EPA, 1987).

1,1-DCE is completely absorbed after a gavage dose and distributes to the liver and kidneys (Jones and Hathway, 1978). Metabolism to end products of alcohols and carboxylic acids involves the formation of toxic intermediates which may interact with tissue macromolecules (Liebler et al., 1984). Renal and biliary excretion is rapid (Jaeger et al., 1977). Humans exposed via inhalation to high concentrations of 1,1-DCE exhibited CNS depression and headaches (NIOSH, 1979). Since much human exposure is to a mixture of chlorinated ethylenes it is difficult to attribute effects specifically to 1,1-DCE.

Animal studies show that the most sensitive toxic endpoint of 1,1-DCE is liver damage, including fatty infiltration, hypertrophy of liver cells, and necrosis (Chieco et al., 1982; Rampy et al., 1977; Quast et al., 1983). No reproductive or developmental effects have been shown to occur with this chemical. 1,1-DCE has been shown to be mutagenic with activation (IARC, 1982; Jacobson-Kram, 1986). A few studies have shown an increase in tumors after inhalation exposure to 1,1-DCE, but no oral exposure studies have demonstrated a carcinogenic effect (NTP, 1982). EPA has concluded that most studies of this chemical do not support a significant, treatment related increase in tumor incidence (EPA, 1987). As a result, CAG classifies it in class C (possible human carcinogen) and has estimated that lifetime ingestion of water (2 liter/day) containing 2.3 ug 1,1-DCE/liter would present an increased cancer risk of 1 excess cancer per population of 100,000. Because of the inconclusive nature of the classification of 1,1-DCE, ODW has set a lifetime health advisory at 7 ug/l for non-carcinogenic endpoints, based on a reference dose of 0.0143 mg/kg/day (drinking water equivalent of 350 ug/l). The toxic endpoint for this recommendation is fatty deposition in the liver (Quast et al., 1983).

Because of the ubiquitous occurrence of lead, and the widespread incidence of exposure, an immense body of literature has accumulated on this metal. It is not within the scope of this document to review all the information available on this subject. A very comprehensive review of the lead literature was conducted by EPA in 1986 (EPA, 1986) and by ATSDR in 1988 (ATSDR, 1988). This review is taken from these reports.

Lead can be absorbed well via inhalation exposure (up to 100%) and ingestion exposure (50% for children and about 15% for adults, depending on the media it is associated with). Dermal absorption is of much less significance. Lead distributes to three compartments; the blood (specifically the erythrocytes and hemoglobin), soft tissue (kidney, liver and brain) and bone (about 95% of the adult body burden, 73% in children). Lead distributes readily to the fetus. Inorganic lead is not metabolized. Any lead not absorbed is eliminated in the feces. Lead stored in bone can be mobilized and redistributed in the body, and therefore chronic, cumulative exposure is of concern.

Low level lead exposure interferes with heme biosynthesis by altering activity of enzymes involved in heme synthesis. This interference in turn affects the synthesis of hemoglobin and cytochrome P-450 and electron transfer cytochromes. The lowest level at which this effect has been observed is 40 ug/dl (EPA, 1986). Low levels of lead may also decrease the circulating levels of 1,25-dihydroxyvitamin D (an active form of vitamin D), which acts in calcium homeostasis in the body. Prenatal exposure to low levels of lead can lead to growth retardation in infants. Lead can also cause a variety of neurobehavioral effects in children at very low exposure levels. There is some evidence that effects may occur in humans below the current CDC guideline of 25 ug/dl. These include cognitive deficits, auditory pathway effects, ALA-D inhibition, pyrimidine-5'-nucleotidase inhibition, and vitamin D inhibition. There is great controversy about the threshold for the cognitive effects. A threshold for neurobehavioral effects from lead exposure has not been determined. The soil guideline commonly used at MDH is 1000 mg/kg in soil.

The PCB mixture found at this site is Aroclor 1254. In several animal studies, intestinal absorption of PCBs was shown to be essentially complete (Albro and Fishbein, 1972; Matthews and Anderson, 1975a), and the Yusho incident demonstrated efficient uptake of PCBs by humans (Kuratsune et al., 1972). In occupational exposure case histories, humans have been shown to absorb PCBs via the skin and respiratory tract. Once absorbed, PCBs are rapidly removed from the blood and stored in liver and muscle (Berlin et al., 1975) and the more highly chlorinated PCBs (such as Arochlor 1254) are redistributed to skin and adipose tissue (Finklea et al., 1972; Kuratsune et al., 1976). PCBs are metabolized in the liver. The degree of metabolism depends on chlorine content and on the site of chlorination (Sundstrom et al., 1976; Lutz et al., 1977). Chlorination also affects lipid solubility, which affects storage of the compound. PCBs stored in human tissue are the more highly chlorinated compounds,

which suggest metabolism and/or elimination of the less chlorinated compounds (Kutz and Strassman, 1976). Only about 20 percent of PCBs with with 6 or more chlorines may ever be eliminated, although 90 percent of PCBs with 5 or fewer chlorines are excreted within 42 days (Matthews and Anderson, 1975b). PCBs are also distributed to the fetus via the placenta, and to the newborn via breast milk (EPA, 1987; Jensen, 1983).

Evaluating the health effects of PCBs is difficult because they are mixtures, and each mixture behaves different kinetically and, therefore, exerts different health effects quantitatively. PCBs have great tendency to bioaccumulate, which makes chronic exposure a great concern. Subchronic and chronic PCB exposure in animals elicits several general effects; skin lesions, immune effects, and alterations to the liver. In many studies, skin lesions are reported as the first sign of PCB intoxication. Skin lesions were seen in monkeys exposed to 2.5 ppm Aroclor 1248 (Allen et al., 1979). Occupational exposure has been associated with skin and mucous membrane effects (ATSDR, 1987). Liver weight increases have been noted in chronic animal studies at doses of 5 ppm Aroclor 1242 and liver lesions and degeneration at higher doses (Bruckner et al., 1974). PCBs have also been shown to induce hepatic liver microsomal enzymes in several species (Litterst and VanLoon, 1972; Allen et al., 1974). PCBs have been shown to affect both cell mediated and humoral immunity (Kimbrough, 1985; Biocca et al., 1975; Thomas and Hinsdall, 1978).

Some fetotoxicity has been noted in infants born to mothers exposed to PCBs by consumption of contaminated fish (Fein et al., 1984; Jacobson et al., 1984; Jacobson et al., 1985). Effects include low birth weight, smaller head circumference, and shorter gestation periods. Contaminated fish consumption by mothers was also correlated with impaired autonomic maturity, increased numbers of abnormal reflexes, and poor visual recognition memory measured in infants. These effects have also been noted in newborns whose mothers were exposed occupationally (Taylor et al., 1984). Many of these studies have confounding factors that limit the interpretation of the results. In a series of studies on rhesus monkeys exposed to Arochlor 1248 in their diet, the transplacental toxicity of PCB exposure was demonstrated. Treated females showed decreased rates of conception, increased fetal death, decreased number of live births, low birth weights, hyperpigmentation, and infant mortality (Allen and Barsotti, 1976; Barsotti et al., 1976). Ringer et al. (1972) showed increased mink kid mortality, which was increased with increased consumption of fish from the Great Lakes (Aulerich et al., 1971), to be a result of PCB contamination in the fish. PCBs have been shown to cross the placenta and cause abortions or fetal mortality in many studies in many species. In some cases, fetotoxicity has been observed without signs of overt maternal toxicity. PCBs are also transferred to rhesus monkey offspring via breast milk, which results in infant mortality and signs of PCB-induced toxicity (Allen and Barsotti, 1976; Allen et al., 1979).

No adequate studies have been conducted to determine if long-term exposure to PCBs causes cancer in human (EPA, 1984), although the evidence of association is suggestive (EPA, 1988). PCB exposure has resulted in an increased incidence of hepatocellular carcinoma in several animal

studies. Data from the most recent study (Norback and Weltman, 1985) were used by EPA as the basis for carcinogenic risk assessment. CAG has classified PCBs in class B2 (probable human carcinogen), and has estimated that lifetime ingestion of water (2 liter/day) containing 0.045 ug/l PCB would present an increased cancer risk of 1 excess cancer per population of 100,000. Fish are a significant source of human PCB exposure (ATSDR, 1987; EPA, 1988).

B. Implications of Media Specific Exposure

1. Ground Water

The contamination of the municipal wells in Waite Park by VOCs was identified in December 1984. The dumping of the chemicals into the ground by EM employees may have begun as early as 1969. Through modeling efforts, it is estimated by MDH staff that the contaminants could have reached the wells quite soon after the contaminants were dumped. It is therefore possible that the people using municipal water in the city of Waite Park were exposed to VOCs from 1970 to 1985, a period of 15 years. At the concentration identified in 1984, persons consuming the water were at possible risk for health effects from ingestion of PCE, TCE and 1,1-DCE, although the risks from ingestion of the mixture is unknown.

B. Surface Water

There does not appear to be PCB contribution to the Sauk River from the BN site. Although metals may be present in the sediments at the BN outfall, metal concentrations are not increased in the water. Therefore, the exposure to any metals from the site in the river sediments are unknown but not likely to be appreciable. There are no indications that this potential exposure pathway is of public health concern.

3. Soil

There is potential for health effects due to lead exposure to children who might come in contact with the sandblast sand piled to the west of Tenth Avenue on the BN site. It is not possible to derive a potential blood lead level from exposure to this media, and therefore it is not possible to define specific potential health effects associated with exposure to the sand. However, given that a threshold for effects has not been determined, the potential for health effects cannot be ruled out.

In addition, it is not known if the oil seeps that appear around the lagoon area in this area contain PCBs; however, PCBs were identified as contaminants in other oily soil samples in the area. If the oil seeps contain PCBs, there is potential for enhanced dermal absorption of this compound if anyone were to contact the oil. It is not possible to determine the health effects of contact with this substance because the dose-response data is not available, but potential health effects from exposure to this oil cannot be ruled out.

4. Air

The model used to predict the chemical emissions concentrations at the Waite Park water supply and at the EM facility is designed to over-predict downwind concentrations of stack emissions at ground level. This model is designed to be conservative; it is extremely unlikely that any measured value would be greater than predicted values (MPCA, 1988). Because of the conservatism and overpredictive nature of the model, the use of modeling is preferred by MPCA to monitoring. The ambient concentrations predicted by the model are well below the concentrations estimated to produce human health effects. Human health should be adequately protected from impacts from the air stripping remediation.

Air-borne lead is a potential source of human health concern; however, at this time there is insufficient information to accurately assess the implications of exposure to possible air-borne lead.

5. Biota

MDH has determined that the level of PCBs in fish in the reach of the Sauk River near St. Cloud is of concern and advises fisherman to limit consumption of those fish to one meal per week if consumed on a short term basis or one meal per month if consumed on a long-term basis. There are no other biota in the river which constitute a significant part of the human diet. As stated in previous sections, BN does not appear to be the source of the PCBs in the Sauk River.

VII. CONCLUSIONS AND RECOMMENDATIONS

Based on information available, exposure to contaminants in the ground water from the municipal water system has been mitigated. The proposed remediation outlined in the forthcoming ROD should protect public health in the future by removing the contaminants from the ground water and the soil at the EM property. Remediation is not expected to pose a threat to human health.

There is potential for health problems to develop as a result of previous long-term exposure to the VOCs in the water system. Although the length of human exposure to the chemicals is unknown, results of modeling estimate that it is possible that length of exposure could have been as long as 15 years.

The area to the west of Tenth Avenue at the BN site poses a potential human health threat because of the presence of sandblast sand which contains a high amount of lead and other heavy metals and which is easily accessible to children playing at the site. There is also a potential human health threat because of the presence of an oily seep which is potentially contaminated with PCBs. Access to these contaminants should be restricted, either by removal of the contaminated media from the area or by restricting access to the area.

The soil and ground-water contamination at the BN site will be addressed with additional FS work at the site.

The City of Waite Park should be advised not to develop the area until remediation is complete.

MDH will return to this site to evaluate new BN data and to track progress on the recommendations here.

Based on the available information, this site is considered to be of public health concern because of the risk to human health caused by past exposure to contaminants in drinking water and the likelihood of exposure to hazardous substances to children in contact with the sandblast sand or the oily waste seeps.

VIII. PREPARERS OF REPORT

Environmental Review:

Elizabeth N. Gerbec, Ph.D., Research Scientist II, Minnesota Department of Health

Forest D. Arnold, Research Scientist II, Minnesota Department of Health

Health Effects Review:

Elizabeth N. Gerbec, Ph.D., Research Scientist II, Minnesota Department of Health

REFERENCES

Albro, P. W. and L. Fishbein. 1972. Intestinal absorption of polychlorinated biphenyls in rats. Bull. Environ. Contam. Toxicol. 8:26-31

Allen, J. R., L. A. Carstens and D. A. Barsotti. 1974. Tissue modifications in monkeys as related to absorption, distribution, and excretion of polychlorinated biphenyls. Arch. Environ. Contam. Toxicol. 2:86-95.

Allen, J. R., and L. J. Abrahamson. 1979. Responses of rats exposed to polychlorinated biphenyl for 52 weeks. I. Compositional and enzymatic changes in the liver. Arch. Env. Contam. Toxicol. 8:191-203.

Allen, J. R. and D. A. Barsotti. 1976. The effects of transplacental and mammary movement of PCBs on infant rhesus monkeys. Toxicology. 6:331-340.

ATSDR (Agency for Toxic Substance and Disease Registry). 1988. The nature and extent of lead poisoning in children in the United States: A report to Congress.

ATSDR (Agency for Toxic Substance and Disease Registry). 1988. Toxicological Profile for polychlorinated biphenyl. Draft for public comment. U.S. Public Health Service.

ATSDR (Agency for Toxic Substance and Disease Registry). 1988. Toxicological Profile for Tetrachloroethylene. Draft for public comment. U.S. Public Health Service.

ATSDR (Agency for Toxic Substance and Disease Registry). 1988. Toxicological Profile for Trichloroethylene. Draft for public comment. U.S. Public Health Service.

Aulerich, R. J., R. K. Ringer, H. L. Seagren and W. G. Youatt. 1971 Effects of feeding Coho salmon and other great lakes fish on mink reproduction. Can. J. Zool. 49:616-626.

Barr Engineering. 1986. Plans/Reports submitted pursuant to Parts IV and V of Exhibit B to request for response action. Electric Machinery Site, St. Cloud, Minnesota.

Barsotti, D.A., R. J. Marlar and J. R. Allen. 1976. Reproductive dysfunction in rhesus monkeys exposed to low levels of polychlorinated biphenyl Food. Cosmet. Toxicol. 14:99-103.

Berlin, M., M. Gage and S. Holm. 1975. Distribution and metabolism of 2,5,2',5'-pentachlorobiphenyl. Arch. Environ. Health. 30:141-147.

Biocca, M., J. A. Moore, B. N. Gupta and J. D. McKinney. 1975. Toxicology of selected symmetrical hexachlorobiphenyl isomers: 1. Biological responses in chicks and mice. Paper presented at the national Conference of Polychlorinated biphenyls. EPA 560/6-76-004. pp. 67-72.

Bruckner, J. V., K. L. Khamma and H. H. Cornish. 1974. Effects of prolonged ingestion of polychlorinated biphenyl on the rat. Food. Cosmet. Toxicol. 12:323-330.

Chieco, P., M. T. Moslen and E. S. Reynolds. 1982. Histochemical evidence that plasma and mitochondrial membranes are primary foci of hepatocellular injury caused by 1,1-dichloroethylene. Lab. Invest. 46:413-421.

Cothern, C. R., W. A. Coniglio and W. L. Marcus. 1985. Techniques for the assessment of carcinogenic risk to the U.S. population due to exposure from selected volatile organic compounds from drinking water via the ingestion, inhalation and dermal routes. Office of Drinking Water. EPA.EPA-570/9-85-001.

EPA (U.S. Environmental Protection Agency). 1986. Air quality criteria document for lead. Office of Health and Environmental Assessment, EPA-600/8-83/028aF-dF.

EPA (U.S. Environmental Protection Agency). 1986. Addendum to the health assessment document for tetrachloroethylene (perchloroethylene): Updated carcinogenicity assessment. EPA/600/8-82-005FA. External Review Draft.

EPA (U.S. Environmental Protection Agency). 1987. Health advisory for 1,1-dichloroethylene. Office of drinking water. March 31, 1987.

EPA (U.S. Environmental Protection Agency). 1987. Health advisory for Tetrachloroethylene. Office of Drinking Water. March 31, 1987.

EPA (U.S. Environmental Protection Agency). 1987. Health advisory for trichloroethylene. Office of Drinking Water. March 31, 1987.

EPA (U.S. Environmental Protection Agency). 1987. Exposure to volatilized water contaminants via inhalation. Importance relative to ingestion. Draft report. Office of Drinking Water.

EPA (U.S. Environmental Protection Agency). 1988. Drinking Water Criteria Document for polychlorinated biphenyl (PCBs). Environmental Criteria and Assessment Office. Cincinnati, Ohio.

Fein, G. G., J. L. Jacobson, S.W. Jacobson, P. M. Schwartz and J. K. Dowler. 1984. Prenatal exposure to polychlorinated biphenyl: Effects on birth size and gestational age. J. Pediatr. 102:315-320.

Finklea, J. et al. 1972 Polychlorinated biphenyl residues in human plasma exposed a major urban pollution problem. Am. J. Publ. Health. 62:645-651.

IARC (International Agency for Research on Cancer). 1982. IARC Monographs on the evaluation of carcinogenic risks of chemicals to humans. Chemicals, industrial processes and industries associated with cancer in humans. IARC Monographs. 1-19 (4): 262-264.

Jacobson, J. L., S. W. Jacobson, P. M. Schwartz, G. G. Fein and J. K. Dowler. 1984. Prenatal exposure to an environmental toxin: A test of the multiple effects model. Dev. Psychol. 20:523-532.

Jaeger, R. J., L. G. Schoner and L. J. Coffman. 1977. 1,1-dichloroethylene hepatotoxicity: Proposed mechanism of action of distribution and binding of ^{14}C radioactivity following inhalation exposure in rats. Environ. Health Perspect. 21:113.

Jones, B. K. and D. E. Hathway. 1978. The biological fate of vinylidene chloride in rats. Chem. Biol. Interact. 20:27-41.

Jensen, A. A. 1983. Chemical contaminants in human milk. Residue Rev. 89:1-128.

Kimbrough, R. D. 1985. Laboratory and human studies on polychlorinated biphenyls (PCBs) and related compounds. Env. Health. Perspect. 59:99-106.

Kuratsune, M., T. Yoshimura, J. Matsuzaka and A. Yamaguchi. 1972. Epidemiologic study on Yusho, a poisoning caused by ingestion of rice oil contaminated with a commercial brand of polychlorinated biphenyl . Environ. Health Perspect. 1:119-128.

Kutz, F. W. and S. C. Strassman. 1976. Residues of polychlorinated biphenyl in the general population of the United States. In: National Conference on Polychlorinated biphenyl . EPA 560/6-75-004.

Liebler, D. ., M. J. Meredith and F. P. Guengerich. 1984. Reactive metabolites of vinylidine chloride: Characterization of covalent adducts formed with proteins and glutathione in microsomes and isolated hepatocytes. Cancer Research 22:5482-5489.

Litterst, C. L., T. M. Farber, A. M. Baker and E. J. VanLoon. 1972. Effect of polychlorinated biphenyls on hepatic microsomal enzymes in the rat. Toxicol. Appl. Pharmacol. 23:112-122.

Lutz, R. J., R. L. Dedrick, H. B. Matthews, T. E. Eling and M. W. Anderson. 1977. A preliminary pharmacokinetic model for several chlorinated biphenyls in the rat. Drug Metab. Dispo. 5:386-396.

Matthews, H. B. and M. W. Anderson. 1975a. The distribution and excretion of 2,4,5,2',5'-pentachlorobiphenyl in the rat. Drug. Metab. Dispo. 3:371-380.

Matthews, H. B. and M. W. Anderson. 1975b. Effect of chlorination on the distribution and excretion of polychlorinated biphenyl Drug Metab. Dispo. 3:371-380.

NIOSH, 1979. C. D. for Occupational Standard.

Nomiyama, K., Nomiyama, H. and H. Arai. 1986. Reevaluation of subchronic toxicity of trichlorethylene. Toxicol. Lett. 31(suppl):225.

Norback, D. H. and R. H. Weltman. 1985. Poly induction of hepatocellular carcinoma in the Sprague-Dawley rat. Environ. Health Perspect. 60:97-105.

NCI. 1976. National Cancer Institute. Carcinogenesis bioassay of TCE U. S. Dept. of Health, Education and Welfare. U.S. Public Health Service.

NCI. 1977. National Cancer Institute. Bioassay of tetrachloroethylene for possible carcinogenicity. U.S. Dept. of Health, Education and Welfare. U. S. Public Health Service.

NTP. 1982. National Toxicology Program. Carcinogenesis bioassay for trichloroethylene. No. 82-1799.

NTP. 1982. National Toxicology Program. Carcinogenesis bioassay for vinylidene chloride (CAS no. 75-35-4) if F344 rats and B6C3F1 mice (gavage study). NIH Publication No. 82-1784.

NTP. 1986. National Toxicology Program. Toxicology and carcinogenesis of tetrachloroethylene (perchloroethylene) (CAS No. 127-18-4) in F344/N rats and B6C3F1 mice (inhalation studies). Natl. Toxicol. Program Tech. Rep. Ser. Issue 311.

Quast, J.F., C. G. Humiston, C. E. Wade, J. Ballard, J.E. Beyer, R. W. Schwetz and J. M. Norris. 1983. A chronic toxicity and oncogenicity study in rats and subchronic toxicity study in dogs on ingested vinylidine chloride. Fund. Appl. Toxicol. 3:55-62.

Rampy, L. W., J. F. Quast, C. G. Humiston, M. F. Blamer and B. A. Schwetz. 1977. Interim results of two-year toxicological studies in rats of vinylidene chloride incorporated in the drinking water or administered by repeated inhalation. Environ. Health. Perspect. 21:33-43.

Ringer, R. K. 1972. Effect of dietary polychlorinated biphenyls on growth and reproduction of mink. 164th Natl. Neet. Am. Chem. Soc. 12:149-154.

Sanders, V. M., A. N. Tucker, K. L. White,B. M. Kauffman, P. Hallett, R. A. Carchman, J. F. Borzellleca and A. E. Munson. 1982. Humoral and cell mediated immune status in mice exposed to trichloroethylene in drinking water. Toxicol. Appl. Pharmacol. 62:358-368.

Stewart, R. D. and H. C. Dodd. 1964. Absorption of carbon tetrachloride, trichloroethylene, tetrachloroethylene, methylene chloride and 1,1,1-trichloroethane through the human skin. J. Ind. Hyg. 25:439-446.

Sundstrom, G., O. Hutzinger and S. Safe. 1976. The metabolism of chlorobiphenyls: A review. Chemosphere. 5:267-298.

Taylor, P. R., C. L. Lawrence, H. L. Hwang and A. S. Patterson. 1984. Polychlorinated biphenyls influence on birth weight and gestation. Am. J. Public Health. 74:1153-1154.

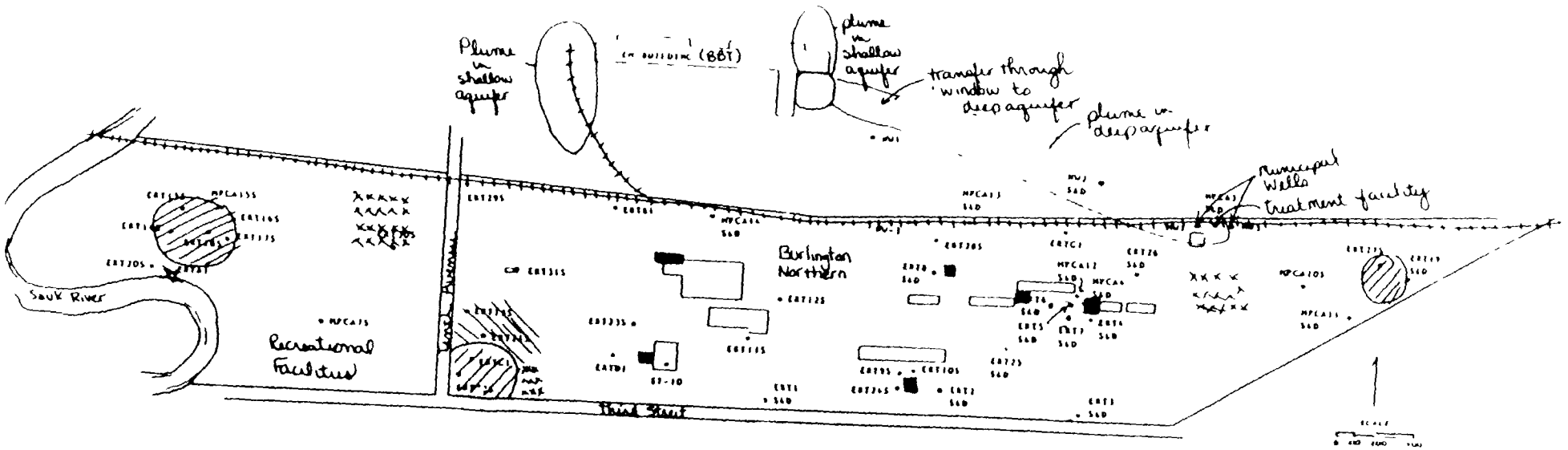
Thomas, P. T. and R. Hinsdill. 1978. Effect of PCBs on the immune responses of Rhesus monkeys and mice. Toxicol. Appl. Pharmacol. 44:41-51.

Vogel T. and P. McCarty. 1985. Biotransformation of perchloroethylene to trichloroethylene, dichloroethylene, vinyl chloride and carbon dioxide under methanogenic conditions. Appl. Environ. Microbiol. 49 (5):166-171.

TABLE 1. Maximum Concentrations of Chemicals Found In Municipal Wells and Monitoring Wells at EM and BN. Units are microgram/liter.

Chemical	Burlington-Northern Deep Aquifer	Burlington-Northern Shallow Aquifer	Electric Machinery Deep Aquifer	Electric Machinery Shallow Aquifer	Municipal Wells
1,1-Dichloroethylene	32	ND	88*	22	94
1,1-Dichloroethane	4	25	290*	240	270
Trichloroethylene	ND	61	54*	5100	60
Tetrachloroethylene	ND	34	340*	15000	680
1,2-Dichloroethylene	24	2	12	4000	11
Chloroform	ND	ND	ND	30	1
Benzene	ND	ND	ND	22	2
1,2-Dichloroethane	ND	ND	ND	240	1
Bromodichloromethane	ND	ND	ND	ND	2
PCBs	ND	5400	ND	ND	ND
Acetone	170	3300	2100*	74	ND

* found in monitoring well between EM and the municipal wells 1 and 3.


Plume in shallow aquifer
(BBT)
plume in shallow aquifer
transfer through window to deep aquifer
plume in deep aquifer
Municipal Wells
treatment facility
Burlington Northern
Sauk River
Recreational Facilities
Third Street
Legend
XX = sand blast grinds
Saygan or oily soil
tank that contaminated soil
CaOH area
VOC contamination at EM site
railroad track
runoff into river
monitoring wells